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Cocoon abdomen – A rare cause of intestinal obstruction



Zia Ur Rehman*

Department of Surgery, The Aga Khan University Hospital, Stadium Road, Karachi, Pakistan

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ABSTRACT

INTRODUCTION: Sclerosing encapsulating peritonitis or abdominal cocoon is a rare condition of unknown etiology in which intestinal obstruction result from encasement of variable length of bowel by dense fibro collagenous membrane.

PRESENTATION OF CASE: A case of young male is reported who presented with features of small bowel obstruction with tender mass in the right iliac fossa. CT scan suggested features of internal herniation. On exploration, he was found to have small intestine, large intestine, stomach and liver covered with a thick cocoon like membrane. The membrane was gently peeled off the small intestine. The patient recovered well and was discharged on an oral diet.

DISCUSSION: Preoperative diagnosis of abdominal cocoon is difficult and most cases are discovered incidentally on laparotomy. Contrast enhanced computed tomography or barium meal may be helpful in preoperative diagnosis. Surgical treatment is the main stay of treatment for this condition. Simple removal of the membrane and lysis of the adhesions produces optimal outcome. Bowel resection is indicated only when the intestine is nonviable.

CONCLUSION: A high index of suspicion and appropriate radiology can prevent 'surprises' and unnecessary bowel resection. Simple removal of the membrane gives a good outcome.

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1. Introduction

The term “abdominal cocoon” was first used by Foo et al. in 1978.¹ It is a rare cause of abdominal pain, weight loss and intestinal obstruction. It is characterized by the total or partial encasement of the small bowel by a fibro-collagenous membrane. The typical patient presents with acute or chronic intestinal obstruction, and/or recurrent episodes of abdominal pain with an abdominal mass. Preoperative diagnosis is very difficult but helpful because it can prevent inappropriate bowel resection. The majority of cases are diagnosed incidentally during laparotomy for the other conditions, as was the situation in this case.

2. Presentation of case

A 31-year-old male presented to us with a history of generalized abdominal pain and abdominal distension for 10 days. The pain was mild to moderate, continuous and associated with nausea and vomiting. Two days earlier he had an ultrasound which showed a loculated ascites and a markedly thickened small bowel

wall. He underwent ultrasound guided aspiration of ascitic fluid which was unsuccessful. His symptoms worsened pain becoming severe. He had no previous medical history of abdominal pain, change in bowel habits or surgery. On examination, he appeared distressed and toxic. His vitals were as follows: pulse 97 beats/min (regular), blood pressure 114/65 mm Hg and respiratory rate 20 per minute. He was afebrile. Abdominal examination showed generalized tenderness more so in the right iliac fossa with a palpable mass. There were no gut sounds. The digital rectal examination was unremarkable. On auscultation, both lung fields had symmetrical breath sounds. An abdominal X-ray showed paucity of bowel gas whereas the erect chest X-ray was normal with no evidence of pneumoperitoneum. Laboratory investigations showed hemoglobin 16.8 gm/dl, hematocrit 50.1%, total leukocyte count 107,000/cc (69.4% neutrophils), platelets 443,000/cc. Serum creatinine and electrolytes were within normal limits. A computer tomography (CT) scan abdomen with oral and intravenous contrast suggested features of internal herniation (Fig. 1). Based on clinical and radiological features decision of laparotomy was made. Exploratory laparotomy revealed 500 ml of yellow serous fluid and a thick white membrane covering stomach, liver, small and large bowel. The greater omentum was not identifiable. Cocoon like membrane was gently dissected off the small bowel. There was well-defined plane of dissection. Care was taken to prevent any serosal injury. The entire small bowel was healthy, with no

* Tel.: +92 21 3493 0051x4751.

E-mail address: Ziaur.rehman@aku.edu



Fig. 1. CT Scan showing dilated bowel loops.

bowel adhesions. Post-operative hospital stay was prolonged due to ileus. Histopathology of the covering membrane revealed chronic inflammation and PCR for tuberculosis was positive. The patient was initially started on oral antituberculous drugs to which he was intolerant so these were later changed to intravenous antituberculous medications. He received peripheral parenteral nutrition and a short course of intravenous steroids to decrease the bowel edema. Nasogastric output gradually decreased and he was discharged on oral antituberculous medications and regular diet. His hospital stay was 20 days. Three weeks later he was readmitted with colicky abdominal pain and vomiting but improved to conservative treatment. He had a good recovery and was well 8 months later.

3. Discussion

Abdominal cocoon can be classified as idiopathic and secondary. The secondary form has been reported in association with chronic peritoneal dialysis, beta blockers treatment (practolol), peritoneovenous shunts, orthotopic hepatic transplantation and abdominal tuberculosis, as was in this case.

The etiology of abdominal cocoons remains unknown. The original causes proposed by Foo et al. were retrograde menstruation and retrograde peritonitis via the fallopian tube.¹ However, these possibilities could not explain the distribution of cases among different ages, genders and geographical locations. D. Magnuire et al. reported five patients in which abdominal cocoon developed after orthotopic liver transplantation.² They suggested that the cocoon

occurred consequent to low grade (subclinical) peritonitis and patients had no significant abdominal signs and did not develop fulminant sepsis.² Persistent low grade inflammation can cause cocoon formation in patients with abdominal tuberculosis which can additionally be characterized by mesenteric thickening; fibrous adhesions and caseous nodules. In the case presented none of these intraoperative features were present.

Preoperative diagnosis is difficult and most cases are discovered incidentally on laparotomy. Contrast enhanced computed tomography or barium meal may be helpful in preoperative diagnosis. Barium meal films may show a characteristic serpentine configuration of the distal dilated small bowel within the cocoon.⁴ On the computed tomography study, “cauliflower sign” or delayed transit might suggest the diagnosis. Bo Wei classified three types according to the extent of encasing membrane³:

Type I – the membrane encapsulates the small intestine partially.
Type II – the entire intestine has been encapsulated by the membrane.

Type III – the entire small intestine and other organs (e.g. appendix, cecum, ascending colon ovaries) are encapsulated by the membrane.

A completely different condition, peritoneal encapsulation may easily be confused with the abdominal cocoon. In cases of peritoneal encapsulation the small and large intestine are seen behind an accessory peritoneal membrane, but the bowel is normal.

Surgical treatment is the main stay of treatment for this condition. Simple removal of the membrane and lysis of the adhesions produces optimal outcome. Bowel resection is indicated only when the intestine is nonviable.

Long term postoperative prognosis is generally excellent.⁴

4. Conclusion

Sclerosing encapsulating peritonitis occurs as a consequence of persistent low-grade peritonitis. Preoperative diagnosis is difficult. A high index of suspicion and appropriate radiology can prevent ‘surprises’ and unnecessary bowel resection. Simple removal of the membrane gives a good outcome.

Conflict of interest statement

None declared.

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Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Key learning point

- Sclerosing encapsulating peritonitis occurs as a consequence of persistent low-grade peritonitis. Preoperative diagnosis is difficult. A high index of suspicion and appropriate radiology can prevent 'surprises' and unnecessary bowel resection. Simple removal of the membrane gives a good outcome.

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